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Letters to the Editor

Pseudomonas pseudomallei-Insulin Interaction

Professor Woods and colleagues provide fascinating laboratory evidence for the suppression of growth of *Burkholderia* (formerly *Pseudomonas*) *pseudomallei* by insulin, both in vitro and in rats (4). They conclude that this is a plausible explanation for the observation that diabetics are especially susceptible to melioidosis, stating that diabetic patients in studies of melioidosis from Thailand and Malaysia were suffering from type 1 diabetes and therefore had insulin insufficiency. Our clinical studies from tropical northern Australia are not consistent with this explanation.

In the 1990–1991 outbreak of melioidosis in Darwin, Northern Territory, 16 (48%) of 33 cases were diabetics, but only one was type 1 (juvenile onset) and only one of the 15 type 2 diabetics (adult onset) was insulin dependent (1). We have now prospectively studied 100 cases of melioidosis since late 1989, and only 2 of the 40 diabetics have been type 1. By far the majority of diabetics have been controlled on diet or oral hypoglycemic drugs, and presumably most are in fact hyperinsulinemic with resistance of target organs to insulin. Almost half of our cases are aboriginal Australians, with an adjusted relative risk of 3.2 (95% confidence interval, 1.2 to 8.8), while the adjusted relative risk for diabetics is 12.9 (95% confidence interval 5.1 to 32.7) (2). Following disruption of traditional lifestyles type 2 diabetes with hyperinsulinemia, obesity, hypertension, and hyperlipidemia is increasingly recognized in aboriginal communities, both urban and rural (3). Further studies will hopefully reconcile the important findings of Professor Woods and colleagues with our clinical data.

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Author's Reply

In regard to Dr. Currie's observations and comments regarding the role of type 1 diabetes in the development of melioidosis, we agree completely with Dr. Currie. In the patient studies which we alluded to, less than 40% of the patients with meliodisis were noted to suffer from type 1 diabetes. Thus, type 1 diabetes is clearly not the only predeterminant for the development of melioidosis, and what we stated in our manuscript was that modulation of the growth of *Pseudomonas pseudomallei* by insulin may influence the pathogenesis of disease due to this organism. We are also aware that not all diabetic patients are type 1, and Dr. Currie's studies indicate that melioidisis occurs in non-type 1 diabetes. Again, we agree that insulin deficiency is not a mandatory prerequisite for the development of melioidosis. We do conclude from our studies, however, that insulin deficiency may serve as an important predeterminant in the development of melioidosis.

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